importance. The adverse effect of sinus disease on the stability of asthma has been described previously in adults, but only recently has this association been documented in children. In children with lower reactive airways disease, more than 50% have sinus radiographs that are abnormal, with a significant minority showing total opacification of one or more of their sinus cavities. The cases of 48 children (mean age 8.2 years) have been described with chronic (more than three months) reactive airways disease and evidence of chronic sinusitis on sinus radiograph; all had been receiving bronchodilators for at least three months with poor control of the asthma, and 18 had received corticosteroids; 14 were nonatopic. With appropriate antimicrobial management, 39 responded both clinically and radiologically. In some children previously requiring daily antiasthmatic medications, including corticosteroids, these medication regimens were able to be completely discontinued following the resolution of their sinus infections. Other investigators have found not only a reduced requirement for antiasthmatic medications, but also a substantially increased responsiveness to bronchodilator therapy.

Because the clinical signs and symptoms of sinusitis can be varied and at times quite subtle, physicians must rely on other diagnostic aids to establish a diagnosis and assess the adequacy of treatment. The sinus radiograph remains the gold standard for diagnosing sinusitis. There is no universal agreement as to how many radiographic views constitute an adequate examination. In children, however, a Waters view may be sufficient. This view clearly shows the maxillary sinuses are involved in about 90% of sinus infections, which may be due to the fact that their ostia are located high on the medial walls where gravitational forces oppose the upward movement of secretions. The Waters view also is useful for showing the frontal sinuses, and some assessment can be made of the ethmoid air cells. The radiographic signs that most closely correlate with positive bacterial aspirates are opacification, air-fluid levels and mucoperiosteal thickening of the mucosal lining. There is always some disagreement as to what degree of mucosal thickening is significant; we feel that at least 4 mm of thickening should be shown to be considered abnormal.

The bacterial characteristics of sinusitis are derived primarily from maxillary antral aspirates and from surgical mucosal specimens. The most common organisms in children with sinusitis are similar to those found in patients with acute otitis media: *Streptococcus pneumoniae, Hemophilus influenzae* and β-hemolytic streptococci; *Branhamella catarrhalis* has been increasingly found in young patients. Furthermore, anaerobic organisms are also commonly cultured when the appropriate media is used. Amoxicillin has proved to be an effective drug and has an 80% cure rate; similar results have been obtained with ampicillin, tetracycline, doxycycline and cefaclor, but penicillin and other cephalosporins are much less effective. Using a combination of erythromycin and sulfisoxazole may be appropriate when amoxicillin treatment fails and there is a suspicion of β-lactamase-producing *B catarrhalis* or *H influenzae*. Under these circumstances, other desirable agents include trimethoprim-sulfimethoxazole and a combination of amoxicillin and clavulanic acid. The appropriate duration of therapy has not been accurately determined, but recent studies indicate that at least three to four weeks are required to effect major radiologic and clinical improvement. If such improvement does not occur after three weeks, it is reasonable to change the antibiotic therapy. If evidence of bronchial hyperreactivity persists in the presence of an unaltered radiograph, antral lavage should be considered; more extensive surgical intervention, such as antral windows, would depend on many variables such as patient age, asthma stability and the location and extent of the disease process.

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Late-Phase Reactions and Chronic Asthma

CHRONIC ASTHMA is characterized by successive attacks of coughing or wheezing, often after respiratory infections. There is usually no obvious correlation to an immediately preceding allergen exposure. Usually minor respiratory irritants, however, such as passive smoking, strong perfume or cooking odors, may cause symptoms. Between attacks, the symptoms may not clear completely, and impaired pulmonary function for airway patency—as determined by the forced expiratory volume in one second (FEV_1), the peak expiratory flow and the maximum midexpiratory flow rate at between 25% and 75% of the values—may persist. Many patients have a few positive immediate skin tests, especially to house-dust mite, cat, pollens and molds, exposure to which does not necessarily correlate with attacks. On provocative inhalation challenge with such an antigen, however, many patients experience a moderate immediate bronchospasm (early phase) that subsides within 30 to 60 minutes, followed in 6 to 8 hours by a more severe bronchospasm, the late-phase asthmatic response (LAR), which is more persistent; these subjects have been called dual responders. Some patients with asthma, however, experience only the early response and others have minimal early but profound LAR. Pretreatment with classic bronchodilators (β-adrenergic agents and theophylline) before challenge can prevent the early response, but is less useful in LAR. Pretreatment with corticosteroids prevent or ameliorate only the LAR, while cromolyn sodium pretreatment blocks both early- and late-phase responses.

Studies in allergic humans and dogs suggest that in many, but not all, allergic persons, the combination of antigen and IgE antibodies on mast cells releases histamine, producing a transient reaction. Such activated mast cells, however, release chemotactic mediators, such as leukotriene B_4 and neutrophil and eosinophil chemotactic factors of anaphylaxis, which attract neutrophils and eosinophils into the inflammatory sites in bronchi and skin. These inflammatory cells release their granular contents, such as neutrophil oxidation products, eosinophil basic proteins that damage the tissues

locally. This tissue damage is responsible for the nonspecific hyperirritability of the airways causing chronic asthma. This has therapeutic implications, for these late-phase reactions respond to corticosteroid and cromolyn therapy, but less well to classic bronchodilators. Patients with asthma who have late-phase reactions may respond better to specific immunotherapy than do those who have only immediate early asthmatic responses. This supports the efficacy of immunotherapy in chronic asthma.

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